AMENDMENTS TO THE SPECIFICATION

Please amend the paragraph beginning at column 23, line 34 as follows:

Measurements of dissociation constants allow different triplets to be ranked in order of preference according to the strength of binding. The examples here indicate that the contacts from either position -1 or +3 can contribute to discrimination. Also, the ambiguity in certain binding site signatures referred to above can be shown to have a basis in the equal affinity of certain [figures] fingers for closely related triplets. This is demonstrated by the K_ds of the finger containing the amino acid sequence RGDALTSHER (Seq ID No. 100) for the [triple] triplets TTG and GTG.

Please amend the paragraph beginning at column 27, line 66 as follows:

Having, established DNA discrimination in vitro, the inventors wished to test whether the anti-BCR-ABL peptide was capable of site-specific DNA-binding, in vivo. The peptide was fused to the VP16 activation domain from herpes simplex virus (Fields 1993 Methods 5, 116-124) and used in transient transfection assays (FIG. 9) to drive production of a CAT (chloramphenicol acetyl transferase) reporter gene from a binding site upstream of the TATA box (Gorman et al., Mol. Cell. Biol. 2, 1044-1051). In detail, the experiment was performed thus: reporter plasmids pMCAT6BA, pMCAT6A, and pMCAT6B, were constructed by inserting 6 copies of the $p190^{BCR\text{-}ABL}$ target site (CGCAGAAGCC) (Seq ID No. 121), the c-ABL second exon-intron junction sequence (TCCAGAAGCC) (Seq ID No. 122), or the BCR first exon-intron junction sequence (CGCAGGTGAG) (Seq ID No. 123) respectively, into pMCAT3 (Luscher et al., 1989 Genes Dev. 1507-1517). The anti-BCR-ABL/VP16 expression vector was generated by inserting the in-frame fusion between the activation domain of herpes simplex virus VP16 (Fields 1993) and the Zn finger peptide in the pEF-BOS vector (Mizushima & Shigezaku 1990 Nucl. Acids Res. 18, 5322). C3H10T1/2 cells were transiently co-transfected with 10 mg of reporter plasmid and 10 mg of expression vector. RSVL (de Wet et al., 1987 Mol. Cell Biol. 7, 725-737), which contains the Rous sarcoma virus long terminal repeat linked to luciferase, was used as an internal control to normalise for differences in transfection efficiency. Cells were transfected by the calcium phosphate precipitation method and CAT assays performed as described (Sanchez-Garcia et al., 1993 EMBO J. 12, 4243-4250). Plasmid pGSEC, which has five consensus 17-mer GAL4-binding sites upstream from the minimal promoter of the adenovirus Elb TATA box, and pMlVP16 vector, which encodes an in-frame fusion between the

DNA-binding domain of GAL4 and the activation domain of herpes simplex virus VP16, were used as a positive control (Sadowski et al., 1992 Gene 118, 137-141).